to the appearance of fever. Doubtless much of this malignancy was due to the combined influence of both affections. While going about, and for some days before confinement to bed, the patient's mind was observed to be impaired and occasionally she was delirious. This delirium continued for six weeks. When she came under our observation at the commencement of the second week of her sickness, the following symptoms were present: The condition of her mental faculties amounted almost to dementia. The pulse was excessively feeble, and not exceeding ninety. Fever of a very low grade, with nocturnal exacerbations, followed by protracted remissions, during which the temperature sank to a very low degree. Her tongue was never coated nor dry at any period; but was as tremulous when an attempt was made to protrude it as that of a person suffering with mania-à-potu. This tremulousness extended to the entire muscular system. Diarrhœa was both troublesome and exhausting. Towards the latter stages emaciation became extreme. The skin became covered with large sloughs and ulcers, probably to the number of thirty. These sloughs formed more particularly about the large points. Some of them measured three inches across. When about to form, a small vesicle appeared under the cuticle, a circular slough of the skin then rapidly formed, and, after separating, left a deep and clean excavation, often extending to the muscles beneath. While these sloughs formed more readily on points exposed to pressure, they appeared indiscriminately over the surface about the large joints, back, &c. They neither granulated nor suppurated until the beginning of convalescence. In this case muscular prostration was extreme. When the patient was propped up in bed occasionally for a few moments, the heart would almost cease to act; the slightest muscular exertion greatly embarrassed her respiration.

Nitric acid, nitrate of silver, occasional opiates, and diffusible stimulants, with a sufficiency of nourishment, were administered, and continued during her entire illness—which was of more than two months' duration—with the exception of about a week, when she took iodide of potassium. The sloughs and ulcers assumed so aggravated a character about that period as to demand some antisyphilitic remedy. Under the iodide of potassium the ulcers improved perceptibly. A further increase of the adynamic symptoms compelled us to suspend this remedy, and again resort to the acid a second time. The patient is now to all appearance convalescing permanently, and, without a relapse, will recover her former health.

ART. III.—*Observations on Bronchial Respiration.* By J. BLOODGOOD, M. D. (Read before the Chicago Academy of Medicine, Nov. 4th, 1859, and published by request of the Society.)

WHEN the ear or stethoscope is applied over the region of the trachea on a healthy subject, the sound produced by the air in its passage through it is distinctly heard, and is recognized as that of tracheal respiration. At a lower position on the upper part of the chest, this sound undergoes some modification in intensity and pitch, approaching more nearly to that of the vesicular respiratory murmur; into which it soon becomes completely merged.

As the aggregate area of the bronchial ramifications is very considerably greater than that of the main trunk, the air of respiration in penetrating the substance of the lungs must move with a constantly diminishing velocity; as the structure of the bronchial tubes throughout is undoubtedly that best adapted for the transmission of air with the least possible friction; as friction of the air upon the walls of the tubes and cells is the element upon which the respiratory sounds depend, and as we have seen that this friction is constantly diminishing from above downwards by reason of diminished velocity, it follows that in a state of health no sound, whether audible at the surface or not, can be generated in the middle or lower portions of the lungs so great as that of the tracheal respiration itself.

In bronchial inflammation, various sounds which have no existence in healthy respiration, are produced. The mucous rhonchus, caused by the bubbling of air through fluid in the bronchial tubes, easily recognized, varying in character according to the size of the tubes, the position, quantity, and consistency of the fluid; the sibilant rhonchus, probably produced by a circumscribed, somewhat circular constriction of a tube, giving rise to a sound more or less resembling a whistle; and the sonorous rhonchus when the sides of a tube are pressed very nearly together, either by inflammatory thickening, by external pressure, or by the presence of tenacious mucus, causing the air to assume sonorous vibrations similar to those produced by the reed of a hautboy or clarionet, and producing sounds varying from that of a snore to the coo of a pigeon, or the hum of a bee. All of these sounds may, with their many varieties, be readily distinguished when present, though the parenchyma of the lung may retain its normal condition, and sometimes without even the necessity of applying the ear to the walls of the chest.

To enter upon an elaborate description of these rhonchi and their various modifications, with the causes which produce them, is foreign to my present purpose, and would be to reiterate what is familiar to every intelligent physician. I merely wish in this connection to call attention to the fact that when the rational symptoms of bronchial disease exist to any extent, wherever located, whether deeply or superficially, the stethoscope seldom fails to discover it by the physical signs, showing that the tissue of the lung, though an imperfect conductor of sound while in a state of health, is yet a sufficiently good one to transmit to the nearest surface any unnatural sound produced within it.

Dr. John Forbes says, what I believe to be true, that

> "When the respiratory sound is perceptible over all its natural sphere and with its natural character, we may be assured that there exists no disease within the chest, which materially affects the lungs; we may be certain, at least, that the air-cells are neither obstructed by fluids poured into them, nor by compression from without. When the natural sound is altered, or no longer exists, we may be assured that disease is present."

This statement is an admission of the fact that any unnatural sound generated anywhere within the chest, either by fluid within the air-cells, or by pressure without, is recognizable at the surface, and if so, any *natural* sound produced there of equal intensity, is recognizable also.

I wish on this occasion to invite especial attention to the form of bronchial or tubular respiration, often heard, because, so far as I have been able to ascertain, it is uniformly attributed to a cause that is insufficient to explain it, one that sometimes exists without the corresponding sound, and is sometimes wanting when the sound itself is present, to wit, the conducting power of hepatized lung which renders audible a sound, usually unheard, but naturally produced by the passage of air to and fro in the bronchial ramifications.

Dr. C. J. B. Williams, in his work on the *Pathology and Diagnosis of Diseases of the Chest*, thus speaks of it :—

> "The second stage of peripneumonia is that in which the lungs present that change in the tissue, which is called by Laennec hepatization. This change consists in the gradual effusion of a semi-solid albumen in the interstitial tissues, which, pressing on and obliterating the cavities of air-cells and smaller bronchi, destroys the spongy texture of the lung, and converts it into a more or less solid mass, such a condition of the air-cells precluding any further ingress of air into them, what stethoscopic signs can we have to indicate this stage of inflammation in the living body? Here still a consideration of the physical state of the organ will teach us to expect, *à priori*, the same phenomena that experience has revealed. We have already had occasion to observe that the healthy lung, from its being composed of conductors of very different powers (air, membrane, and liquid), is a bad conductor of sound, and is therefore incapable of transmitting to its surface slight sounds, remote in the interior. But now that the tissue is rendered more uniformly dense by hepatization, it becomes a better conductor and transmits a sound (usually unheard) of the air passing to and fro in the larger bronchial ramifications. This is the *bronchial* or *tubular respiration* of Laennec and Andral, and marks the advance of the second stage of pneumonic inflammation. This sound when once heard cannot be mistaken; it resembles that produced by blowing through a crow's quill, and is frequently so loud as almost to amount to a whistle, acute and defined it forms a remarkable contrast to the dull diffused sigh of vesicular respiration. In other parts of the lung, if the disease is extensive, the breath sound is puerile."

Dr. Forbes, in the article Auscultation, *Cyclopedia of Practical Medicine*, bears similar testimony. He says :—

> "It was stated that the peculiar sound produced by the passage of the air in the bronchi, termed bronchial respiration, is not audible in the state of health, except occasionally over the track of the very largest branches; no doubt, this sound always exists in the bronchial tubes scattered throughout the lungs, but in the state of health it is rendered imperceptible, partly on account of the bad conducting powers of the spongy texture of the lungs, and partly from its intermixture with the more general sound caused by the expansion of the air-cells. When, however, the vesicular respiration is obstructed by disease, either by the obstruction or compression of the air-cells (as in inflammation of the lungs or pleura), we then hear distinctly the characteristic sound of bronchial respiration, even in the tubes of middling calibre. This variety of respiration becomes, therefore, an important sign in certain diseases."

Dr. Watson, *Practice of Physic*, says :—

"Sometimes, when this crackling ceases," alluding to the fine crepitation of the first stage of pneumonia, "the ear applied to the corresponding surface of the chest feels it heave up in inspiration, but catches no sound at all. Much more commonly, however, a new sound reaches the ear. It is not the vesicular rustle, it is not the minute crepitation, but a whiffling sound is audible like that produced by blowing through a quill. This is the sound to which the term bronchial respiration has been given, and the name expresses well the fact. I mentioned before, that in the healthy state we do not hear the air pass through the larger bronchi during inspiration and expiration. The sound, doubtless, is made, but it is obscured and hidden by the smooth rustle of the vesicular breathing which comes from the spongy lung surrounding the large divisions of the bronchi, and intervening between them and the ear; but that spongy structure is now filled up. The hepatized lung permits air to pass through the larger bronchi which are still patent, but it admits none into the vesicles or smaller tubes. It crepitates not when pressed between the thumb and finger; in fact, it is converted into a solid substance, and conducts the sound in the living body as any other solid substance would do, and, therefore, the whiffling, blowing, gusty sound of the breath, as it enters and departs from the larger bronchial tubes which still remain open, is conveyed to the ear and bronchial respiration is heard."

Again—

"The altered condition of the organ gives rise to altered sounds. Instead of the vesicular breathing of health, or the minute crepitation belonging to the first stage of inflammation, we either hear no sound at all, though we feel the chest heave up against our ear, or we hear what I described under the denomination of bronchial respiration; that is to say, a blowing sound which is conveyed to the ear from the larger and still pervious branches of the bronchi through the solid portion of lung around them, and through the solid walls of the chest. This is what the listener hears when the patient breathes, and when he speaks his voice is heard much more resonant than is natural; much more resonant than in the corresponding spot on the opposite side of the chest. We thus become acquainted with two entirely new sounds which are never heard in a healthy state of the lungs, *bronchial respiration* and *bronchial voice*, or *bronchophony*."

Still again—

"But I say sometimes we hear these morbid sounds in the case in question, and sometimes we hear no sound at all during the breathing. How is that? Why do the existence and degree of the bronchial respiration and bronchial voice vary according to the place and extent of the inflammation? These morbid sounds are most plainly marked when the number and size of the bronchial tubes involved in the hepatization are the greater. They are most distinct, therefore, when the inflammation occupies the upper part of the lung or the central parts, what are called the roots of the lungs, and when it extends thence to the surface; but when the lower portions alone are inflamed, or the inflammation is merely superficial or partial, they may not be heard at all. Again, if the inflammation should be so general and complete as to prevent the chest on the affected side from expanding you will in that case hear no bronchial respiration, for the air in the large bronchi must be stagnant; *bronchophony*, however, may remain."

Andral's views differ slightly from those attributed to him in the passage quoted from Dr. Williams. He thinks that the air, being prevented during hepatization from entering the vesicles, strikes with greater force than in healthy respiration against the sides of the tubes, and thus elicits a louder sound.

That hepatization is by far the most frequent cause of this peculiar respiratory sound is doubtless true. Dr. Walshe says: "The tubular form occurs in perfection in but one condition of lung, that of hepatization; so true is this that tubular and pneumonic breathing may be used as convertible phrases." That hepatization, by rendering the lung a better conductor, enables it to transmit to the surface sounds produced within it, otherwise inaudible, is also true; but that a sound so loud as almost to amount to a whistle, acute and defined, often of greater intensity than any other *naturally* produced within the chest, louder than the various bronchial rhonchi resulting from disease, which, when present to any extent, are always audible, whether the intervening lung be hepatized or not, can be naturally generated there by the passage of air to and fro in the bronchial ramifications, and yet be heard only through the conducting power of hepatized lung requires proof. The theory seems to be entirely inadequate to explain the fact.

All the respired air passes through the trachea where all the conditions that the respiratory organs furnish in health for the production of sound exist in their greatest perfection, and where all the sound produced, which is never so acute as almost to amount to a whistle, can be transmitted to the surface under the most favourable circumstances for being heard without the intervention of lung or any other non-conducting tissue to obstruct its passage. As the conditions necessary for the production of sound are constantly approaching more and more nearly from above downwards to those of the vesicles themselves, so must be also the sound, which can never be so loud below as that heard in the trachea, unless an effect can be greater than the cause that produced it. Every one here knows too well the objects to be accomplished by the function of respiration to believe that the mere production of sound is one of them.

The reason given by Dr. Forbes, in part, and by Dr. Watson, in full, for the absence of bronchial respiration in health, that it is obscured and hidden by the smooth rustle of the vesicular breathing is quite insufficient. No dull diffused sigh can annul a sound of higher pitch and greater intensity, that but for its presence would be heard; a swarm of bees, however large, never drowns the music of the tin pan that is put in requisition for the purpose of hiving them; and supposing the dull sound could conceal the other, what explanation can be given of the fact stated that in hepatization we sometimes hear no sound at all, though we feel the chest heave up against our ear?

How can the chest heave up against the ear unless it expands? How can it expand unless air enters it? How can air enter it without passing through the bronchial tubes! and how pass through those tubes without developing bronchial respiration if the conditions necessary to its development are simply the passage of air and the withdrawal of the confusing vesicular sound, particularly when the only other condition claimed to be necessary, the greater conducting power of hepatized lung is added thereto? All the necessary conditions are present here, but the legitimate effect is absent. If it be true that bronchial respiration is merely the natural sound of transmission, rendered audible by the absence of the vesicular murmur or the presence of hepatization, one or both, the latter, however slight, if sufficient to destroy the vesicular sound, ought to develop the other in all cases not sufficiently extensive to exclude air from the lung altogether. Dr. Watson tells us that when the inflammation is so general and complete as to prevent the chest on the affected side from expanding, we shall hear no bronchial respiration, the air in the larger bronchi being stagnant, while bronchophony may remain. How can this be if both are naturally produced within the chest and rendered audible by the same cause? The object to be accomplished in speaking is to create sound, the very essence of speech, by creating certain vibrations in the larynx which are communicated to the column of air in the bronchial tubes, transmitted to the surface and recognized there as vocal resonance in health. When rendered more audible by hepatization it is still manifestly only an exaltation of the same natural phenomenon, not an entirely new sound, as Dr. Watson asserts. The object in breathing is the decarbonization of the blood, in which process sound is useless, and forms no part more than what necessarily attends the introduction of air to the blood.

If the sound called bronchial respiration were a necessary part of that process, it should be as readily heard through the hepatized lung of the affected side as the bronchophony, which originates in the same place and is heard.

In healthy respiration the amount of air required for each act of inspiration is proportionate to the space to be filled. When the space is diminished by hepatization the quantity required will also be diminished correspondingly. If the right lung, for instance, is wholly hepatized, the left lung will receive very little, if any, more air at each inspiration than it did in health; the power that causes the air to enter being atmospheric pressure, always uniform, and the expansive force of the respiratory muscles often diminished rather than increased during the inflammatory state. As all the air that enters the lungs is drawn in to fill the vacuum occasioned by the expansion of the vesicles, the draught must be diminished by any degree of hepatization, and cease entirely when the hepatization becomes complete. The theory of Andral, therefore, cannot be true that the air, being prevented from entering the vesicles, strikes with greater force against the sides of the tubes, for all that causes it to enter a tube at all is the demand created for it in the vesicles which the tube supplies.

The instinctive feeling of want of breath is, doubtless, ultimately connected with the presence of black blood in the pulmonary capillaries, and as in inflammation, the rapidity of the circulation is increased, so must be the frequency of the respiratory acts also; but increased frequency does not necessitate increased force. We are still left without an explanation of any respiratory sound more marked than that of puerile respiration.

A theory that fails to explain the facts sought to be accounted for, or that is in conflict with any of them, cannot be true. Some other explanation, therefore, must be sought.

We have seen that in healthy respiration both the friction and consequent sound produced by the passage of the air are constantly diminishing from the trachea downwards, and, very probably, have admired the ingenuity displayed in bringing the very delicate tissue of the lungs into free contact with the atmosphere without endangering its integrity. In disease the conditions are changed; a small bronchial tube that naturally produces little or no appreciable sound may then become a powerful instrument of it, for sound is not determined by the quantity of matter in motion, nor by the velocity alone, but by the resistance offered. The wind whistling through a keyhole is much more noisy than a far larger quantity passing through the open door. The boatswain's whistle is heard above the storm, and the mountain brook babbles boisterously on its way to the deep but silent river. When a portion of lung becomes hepatized the bronchus that supplied it no longer transmits air to the vesicles below, and can, therefore, no longer produce the sound of transmission, great or small; but as the tube itself remains open by reason of the elasticity of its walls, *the air coming from above having no room to enter except by expelling an equal quantity from it, plays around its mouth in passing downwards, and causes the air within to vibrate like the instrument known as the Pandean pipe*, and thus produces the tubular sound which will be high or low, loud or weak, according to the calibre and depth of the pervious portion of the tube, and the force of the respiratory act, which latter will be greater when the upper and middle lobes are affected than when the disease is confined to the lower portions on account of the greater velocity of the air in those parts. When the tube itself becomes filled with solid or liquid matter, or closed by external pressure, this sound will cease.

This theory explains not only the occurrence of bronchial breathing in pneumonic inflammation, but its occasional absence in that disease, by the tubes themselves becoming filled up, and also its presence sometimes in states of disease in which no hepatization exists. In the summer of 1846, I attended a patient suffering with acute bronchitis, in whom I was surprised to find, on one occasion, under the left breast, a loud, distinct, and perfectly characteristic tubular respiration, which did not exist on the previous day, and which disappeared before my visit ended. The case was one of but moderate severity, no other sign or symptom of pulmonary inflammation existed, and the speedy disappearance of this one would have forbidden such a supposition. It could only be attributed to the temporary presence of a pellet of tough mucus, plugging up a tube at some distance below its mouth. The views here presented begin to bear date from the occurrence of this case.

This theory also explains the development of bronchophony at times, without the corresponding bronchial respiration. Bronchophony being the sound naturally produced in speaking, by the propagation of the vocal vibrations downwards, and known in health as vocal resonance, must necessarily become more distinct by any improvement in the conducting power of the intermediate lung, and may be heard even though an entire side be hepatized, and its tubes be filled up with solid or liquid matter; which latter fact puts an end to bronchial respiration. But if the tubes contain air, it cannot be stagnant, as Dr. Watson asserts, but, communicating freely with the larynx, must receive and transmit the vocal vibrations as perfectly as does the column on the other side, if not more so, being stationary, after the manner of a speaking-tube or stethoscope, in which the column is not stagnant, though it does not move to and fro therein. Under these circumstances, bronchophony will acquire its greatest intensity, and, with all deference to Dr. Watson, bronchial respiration will be produced, because the air cannot pass over the mouth of the bronchus of the diseased side, on its way to the healthy lung, without causing vibrations of the air within it, which will elicit a louder sound than a much larger quantity of air passing freely through the tube would do. This may be easily illustrated with a tube three or four inches long and half an inch in diameter, by blowing forcibly through it to represent the transmission sound, then closing its lower extremity and blowing gently over its mouth, by which a loud tubular sound will be produced with very little effort. Every variety of pitch may be obtained by using a cork for a stopper, and by varying its position in the tube; but it is necessary to its production, that the tube should be perfectly closed. The first case in which my attention was seriously drawn to the value of stethoscopic signs, was one of this kind. The whole right lung was hepatized, and, consequently, immovable, with bronchial respiration most distinct and unequivocal over all its upper part. I have since seen other cases, though fortunately not many, with the same character and the same sign.

Having taken exceptions to Dr. Watson's statement that, "when the entire lung is hepatized, you will have no bronchial respiration, for the air in the larger bronchi must be stagnant," I will quote, in support of my position, from a case recorded in *Andral's Medical Clinic*—"Diseases of the Chest"—than which no higher authority can be found:—

"CASE II. Post-mortem. Red hepatization of the three lobes of the right lung." "This is the first instance we meet of hepatization of the entire of a lung. When the patient entered La Charité, the signs furnished by auscultation and percussion announced the existence of this hepatization, not yet complete, in the middle and lower lobes; the upper lobe was, as yet, but engorged. The following days, it became rapidly hepatized; but, in this lobe, the hepatization was announced by the *bronchial respiration*, whilst in the other lobes—whose alteration, however, seemed to be the same—this characteristic sound was not heard. We think it right, carefully to note these differences of signs afforded by identical lesions, in order that we may be accustomed to recognize these lesions whatever may be the shades announcing them."

This case demonstrates, what any number of negative facts cannot disprove, that the transmission theory is not only unnecessary, but wholly inadequate to explain the existence of bronchial respiration, for it occurs here under circumstances which would render its occurrence impossible, if the theory were true, and which, Dr. Watson says—more in deference to theory than to fact—do render it impossible. The lung is solid and immovable, no air is transmitted through its tubes, and no sound of transmission can by possibility be produced, yet the bronchial respiration is distinctly heard, and must, therefore, be due to a different course.

The phenomena of cavernous respiration and pectoriloquy illustrate this subject. Tubercle is deposited in the capillary structure of the lungs, and does not usually obstruct the bronchial branches, except by external pressure. When it softens and is discharged, an ulcerated cavity remains, the walls of which become sufficiently indurated by the deposition of lymph to prevent collapse and render them impervious to air, except through the opening into the adjoining bronchus, through which the tuberculous matter has been discharged. Here the conditions are essentially the same as in the former case. The air does not circulate through the cavern, but vibrates within it, and develops cavernous respiration and also pectoriloquy —more or less perfect, according to the form and size of the cavity—by *consonating* with the vocal vibrations in the larynx. A small tubercular cavity, with a wide mouth, might generate only bronchial respiration (from which the cavernous differs but in degree) and bronchophony; while a dilated bronchus, with a contracted mouth, might equally well develop cavernous respiration and pectoriloquy, though such combinations may rarely occur. In hepatization of an entire lung, the main bronchus of that side becomes, in effect, a cavern, from which it differs only in form, and *generates*, by the vibratory motion communicated to the air within it by the passing air without, the true tubular respiration, which, we have seen, could have had no existence during a state of health.

I grant that, in certain cases, the natural sound of transmission may become audible through the intervention of hepatized lung—when, for example, a portion of the upper or middle lobe, lying between the tubes that supply the still healthy lower lobe and the surface, is in that state—but the sound will be no louder than the vesicular murmur that obscured it, and, in a majority of cases, will be disguised by, or lost in, the louder bronchial sound produced in the tube that formerly supplied the hepatized part. When the lower lobes are exclusively the seat of hepatization, the transmission theory furnishes no explanation whatever of the bronchial breathing. The tubes that supply them transmit no air, and, according to this theory, must be silent; while those above are still surrounded by healthy tissue, and must transmit the natural, or, at most, the puerile sound. Indeed, if this theory be true, I see not how the bronchial respiration can ever be developed, unless the lower portions remain pervious while hepatization exists above; and, as inflammation and its consequences are much more frequent in the lower than in the upper lobes, bronchial respiration should be the exception rather than the rule, as it manifestly is.

The theory herein presented is sufficient to account for its presence whenever it exists, and for its absence when it is wanting; for its greater frequency and intensity when inflammation affects the upper lobes than when confined to the lower portions; and, if true, will justify sometimes a more favourable prognosis in cases of bronchial respiration than would be warranted if hepatization were present.

ART. IV.—*On the Seat of the Vesicating Principle of Lytta Vittata.* By JOSEPH LEIDY, M. D., Prof. of Anat. in the Univ. of Penna. (Read before the Biological Department of the Academy of Natural Sciences, September 5, 1859.) With a wood-cut.

IT often occurred to me that the vesicating principle of the blistering fly, *Cantharis*, *Lytta*, etc., was the product of some special organ in the insect, and was not diffused through the body of the animal. In polyps, bees, wasps, mosquitos, etc., we find the irritating agencies or poisons produced by particular cells and glands, not to mention the poisons and odorous principles found in special organs of higher animals. A few days since, observing numerous individuals of Lytta vittata feeding on Amaranthus albus, I collected a number, and at a leisure moment experimented with the different parts of the animal to ascertain if the vesicating principle was confined to some special part of the insect. I did not then recollect of having read of others who had performed similar experiments with the same object, but on examination, find in Pereira, under the head of Cantharis vesicatoria, the following remarks.

"The active and odorous principles of cantharides reside principally in the sexual organs of the animals. Both Farines and Zier tell us that the soft contain more active matter than the hard parts. It appears, also, that the posterior is much more acrid than the anterior portion of the body; and Zier says the ovaries are particularly rich in this active matter."

Though I have long been familiar with the researches of Audouin on the anatomy of the Spanish fly, I remembered that he had made no mention of the source of irritating power in the insect. The anatomy of Lytta vittata is so nearly like that of the Cantharis vesicatoria as described by Audouin, that I shall say nothing about it except in the way of reference.